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Gastric myoelectrical activity and intracranial hypertension

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A Study Design
B Data Collection
C Statistical Analysis
D Data Interpretation
E Manuscript Preparation
F Literature Search
G Funds Collection

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Summary

Background: The aim of the study was to evaluate altered patterns of gastric myoelectrical activity in patients with high intracranial pressue due to severe craniocerebral injury producing intracranial hematomas, or to neoplastic processes in the central nervous system.

Material/Methods: The study population consisted of 50 patients admitted to the Department of Neurotraumatology, Jagiellonian University College of Medicine. The controls were 16 healthy volunteers matched for gender and age. Gastric myoelectrical activity was recorded with standard cutaneous electrodes using the Synectics (Sweden) system of data storage and analysis.

Results: The most significant changes in gastric myoelectrical activity were observed in patients after severe head injury with increased intracranial pressure (Glasgow Coma Scale score 4–7). The percentage of bradygastria increased to 46.5 in these patients, and the signal amplitude was also increased. Significant dysrrhythmias occurred in patients with increased intracranial pressure due to brain tumours. The percentage of tachygastria increased to 36.5.

Conclusions: The greatest changes in gastric myoelectrical activity were found in patients with increased ICP and coma (GCS score 4–7) due to head injury. Bradygastria was found in 46.5% of cases.

key words: **bradygastria • tachygastria • intracranial pressure (ICP) • intracranial hematomas • brain tumors**

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BACKGROUND

Patients with increased intracranial pressure (ICP) due to traumatic brain injury (TBI), or to growth processes in the central nervous system, show autonomic dysfunctions manifested by altered gastric myoelectrical activity, which impairs gastric motility. In our study, electrogastrography was applied, as a non-invasive technique for evaluating gastric motility and its disorders. Brain CT was performed to assess intracranial pressure, taking into account the following parameters:

- shift of the cerebral midline;
- the presence of compression of cisternae at the base;
- subarachnoid grooves on the brain vault;
- mass effect.

MATERIAL AND METHODS

The study population consisted of 50 patients with symptoms of elevated ICP, admitted to the Department of Neurotraumatology in the Institute of Neurology aqt the Jagiellonian University College of Medicine between 1999 and 2000.

Table 1 summarizes the demographic characteristics of the patients with elevated ICP.

The symptoms of elevated ICP were produced by traumatic brain injury in 24 patients, and by neoplastic growth in the central nervous system also in 24 patients. In two patients, increased ICP was associated with internal non-communicating hydrocephalus (Figure 1).

In all patients, both with TBI and brain tumor, ICP was measured using computerised tomography (CT), and magnetic resonance imaging (MRI) was additionally used in tumor patients. The following parameters were taken into account:

- shift of the cerebral midline;
- the presence of compression of the third ventricle and cisterns at the base,
- mass effect.

The patients' clinical status was evaluated on the basis of the Glasgow Coma Scale (GCS), developed by Jannet, Murray and Parker, together with neurological parameters, CT and MRI results.

Table 2 shows the GCS scores in the study population.

The GCS evaluates consciousness on the basis of three parameters (eye opening, verbal output and motor responses), each rated on a scale from 1 (no response) to 4, 5, or 6 (normal response in eye opening, verbal output and motor responses respectively), so that the minimum score of 3 indicates no response (profound coma), and the maximum score of 15 indicates unimpaired consciousness.

The patients with elevated ICP were divided into three subgroups:

Group 1: 23 men and one woman, from 24 to 71 years in age, including 18 subjects with intracranial hematomas (6 epidural, 6 subdural, 6 intracerebral) and 6 patients with cerebral contusion and edema resulting in intracranial shift. All patients with severe head injury were comatose (GCS score 4–7). They had the symptoms of an 'autonomic storm', with pulse rate increased to 100–140 bpm, respiratory rate 30–36 per minute, and increased body temperature to 39–41°C. They responded to pain and showed divergent eyeballs, increased limb tonus, deep reflexes and bilateral Babinski's sign. All patients underwent brain CT, which revealed mass effect with signs of ICP and cerebral midline shifted more than 12 mm. Non-invasive EGG was performed on the first or second day after head injury. Enteral feeding was not used in comatose patients after severe head injury within the first 48 hours.

Group 2: 10 men and 6 women, from 18 to 68 years in age, with supratentorial neoplastic growth within the central nervous system. These patients were conscious and responsive (GCS score 10–15). Clinical manifestations of increased ICP, confirmed by brain CT, included headache, bradycardia 40–50 bpm, somnolence, bradypsychia and motor sluggishness. Papilloedema was observed in four patients. The symptoms of elevated ICP developed within 2 weeks to 10 months. All patients underwent CT scans of the head, and four patients additionally MRI. In this group electrogastrography was performed interprandially.

Group 3: 6 men and 4 women, from 37 to 70 years in age, with brain tumor, chronic subdural hematoma, or internal hydrocephalus. These patients were receiving osmotic agents before elective surgery. Papilloedema was not found in this group of patients. Two patients with symptoms of internal hydrocephalus, selected for Pudenz atrioventricular valve placement, had cerebrospinal fluid pressure of 25 cm H_2O measured in the vertebral canal.

Figure 2 shows the mean age in the study groups.

Table 1. Demographic characteristics of patients with elevated intracranial pressure.

Gender	N	%	Age (years)	
			x±SD	min–max
Men	39	78	44.4±14.3	18–71
Women	11	22	56.9±11.4	33–70
Total	**50**	**100**	47.3±14.6	18–71

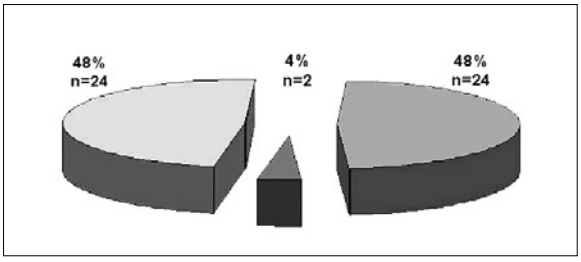

Figure 1. Causes of elevated intracranial pressure in the study population.

Table 2. Glasgow Coma Scale (GCS).

Eye opening	Score	Verbal response	Score	Motor response	Score
Spontaneous	4	Oriented	5	Obeys	6
To loud voice	3	Confused. disoriented	4	Localizes	5
To movement	2	Inappropriate words	3	Withdraws (flexion)	4
Nil	1	Incomprehensible sounds	2	Abnormal flexion posturing	3
		Nil	1	Extension posturing	2
				Nil	1

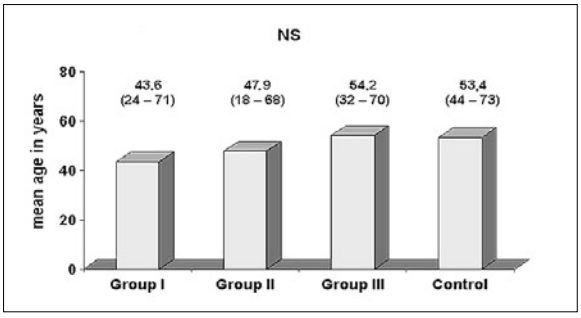

Figure 2. Mean age in the study groups.

The control group consisted of 16 healthy volunteers matched for gender and age without head injury or pathology in the central nervous system.

All subjects underwent one-hour EGG using standard electrodes (Ag/AgCl gel electrodes, Menos, Poland) and digitrapper (Synectics Medical, Sweden). Gastric myoelectrical activity was recorded for 60 min in the fasting state. The signal was digitally processed and computer analyzed. Final analysis was carried out in the Department of Pathophysiology at the Jagiellonian University College of Medicine. Figure 3 shows normal 3D EGG.

Gastric myoelectrical activity was compared between patients with elevated ICP and the controls using the Mann-Whitney test. The same test was applied to compare the subgroups of patients with papilloedema. Analysis of variance was used to compare the four groups (3 groups with elevated ICP and the control group) with respect to gastric myoelectrical activity. If the result was statistically significant, Duncan's multiple comparison test was used to determine where the differences occurred. The Chi squared test was also used to analyze gastric myoelectrical activity as a descriptive variable in the study groups. A p value less than 0.05 was considered statistically significant.

RESULTS

Table 3 summarizes gastric myoelectrical activity in the patients with elevated ICP and the controls.

Bradygastria in patients with elevated ICP was higher by an average of 18% than in the controls ($p<0.02$, Table 4). Normogastria in the study patients was 57.8% on the average, significantly lower ($p<0.001$) than in the controls. Tachygastria in patients with elevated ICP

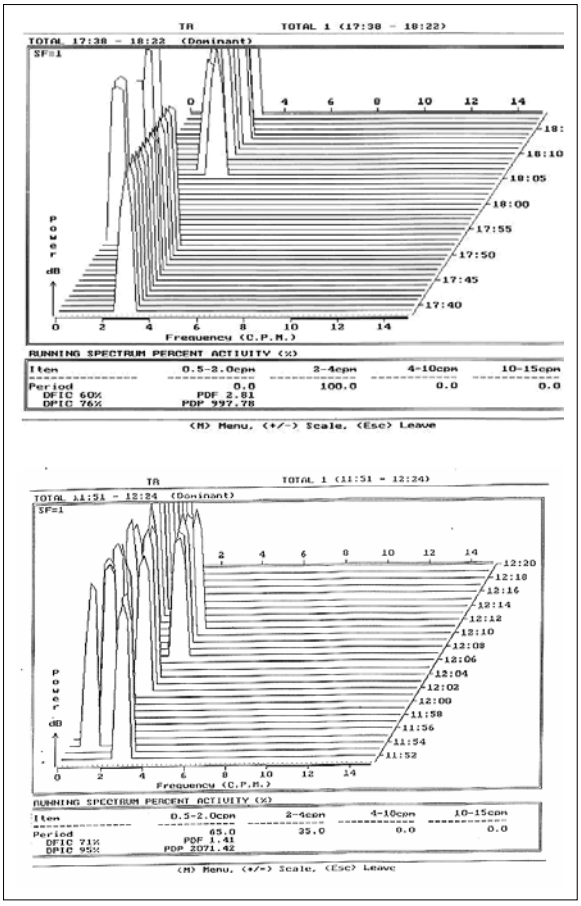

Figure 3. 3D EGG showing normal and abnormal (bradygastria 65% of the recording time).

was 12.9% on the average, and differed significantly from the controls.

Table 4 summarizes the mean GCS scores in the study patients with elevated ICP. The differences are statistically significant ($p<0.001$).

Table 5 summarizes bradygastria in the study groups. The highest bradygastria values were observed in group 1: 46.7%, significantly higher than in group 2 (16.3%), group 3 (4.9%), and the controls (9.7%). (Table 5).

Table 6 summarizes normogastria values in the study groups. The highest percentage of normogastria was observed in group 3 and the controls: 89.2% and 88.3%,

Table 3. Comparison of gastric myoelectrical activity in patients with elevated intracranial pressure and in controls.

Slow wave signal	Group	n	% recording time x±SD min–max	P
0.5–2 cpm	Patients with elevated ICP	50	28.5±26.4 (0–100)	
	Controls	16	9.7±6.4 (0–19.6)	p=0.02
2–4 cpm	Patients with elevated ICP	50	57.8±23.3 (0–100)	
	Controls	16	88.3±7.6 (78–100)	p<0.001
4–10 cpm	Patients with elevated ICP	50	12.9±16.9 (0–66.7)	
	Controls	16	2.0±3.9 (0–12)	p=0.02

Table 4. Mean GCS scores in the study patients with elevated intracranial pressure.

Group	N	GCS x±SD	GCS min–max	P
2	24	5±1	3–7	p<0.001 I#II#III
2	16	13±1	10–15	
3	10	14±1	13–15	

respectively, as compared to 47.2% and 54% in groups 1 and 2, respectively. The differences between group 3 and the controls as compared to groups 1 and 2 was statistically significant at p<0.001.(Table 6).

Table 7 shows that the tachygastria values are the highest in group 2 (29%) and significantly higher (p<0.001) than in group 1, group 3, and the controls. In the 4 patients with elevated ICP and papilloedema, gastric electrical activity was slowed to 56.1% (Table 9). Exclusion of these patients from group 2 brought tachygastria values to 36.5%, but did not affect the level of statistical significance.(Table 7).

Figure 4 shows gastric myoelectrical activity in all study groups, with characteristic high values of bradygastria in group 1, tachygastria in group 2, and normogastria in group 3 and the controls.

Tables 8 and 9 describe group 2, broken down in to patients with and without papilloedema. Those with papilloedema have significantly higher bradygastria time (p=0.004) and significantly lower tachygastria time (p=0.004) as compared to patients in whom no papilloedema was found. The presence of papilloedema did not affect significantly the level of consciousness assessed by the GCS.(Tables 8 and 9).

In group 1, consisting of patients with severe head injury, 22 patients (91.7%) showed predominantly bradygastria, whereas two patients (8.3%) had tachygastria (Table 10).

Table 5. Percentage of bradygastria (0.5–2 cpm) in the study groups.

Group	N	Bradygastria x±SD	Bradygastria min–max	P
1	24	46.5±21.8	22.8–100	p<0.001 I=II=C I#(II.III.C)
2	16	16.3±22.1	0–63	
	*12**	*4.7±7.1*	*0–23.1*	
3	10	4.9±6.2	0–15	
Controls	16	9.7+6.4	0–19.6	

* for group 2, excluding 4 patients with papilloedema

Table 6. Percentage of normogastria (2–4 cpm) in the study groups.

Group	N	Normogastria x±SD	Normogastria min–max	P
1	24	47.2±20.4	0–77.2	p<0.001 I=II III=C **(III.C)#**(I.II)
2	16	54.0±13.7	30–74.1	
	*12**	*57.8±12.1*	*33.3–74.1*	
3	10	89.2±12.9	70–100	
Controls	16	88.3+7.6	78–100	

* for group 2, excluding 4 patients with papilloedema

Table 7. Percentage of tachygastria (4–10 cpm) in the study groups.

Group	N	Tachygastria x±SD	Tachygastria min–max	P
1	24	5.5±11.8	0–50.6	p<0.001 I=III=C **II#**(I.III.C)
2	16	29.0±17.1	0–66.7	
	*12**	36.5±12.1	22.2–66.7	
3	10	4.9±6.2	0–15	
Controls	16	2.0+3.9	0–12	

* for group 2, excluding 4 patients with papilloedema

In group 2, consisting of patients with neoplastic growth within the central nervous system, predominantly tachygastria was observed in 12 patients (75%). In group 3, receiving osmotic agents, eight patients (80%) showed predominantly normogastria, one patient bradygastria, and one patient dysrhythmia (equal times for brady- and tachygastria).

The chi-squared test revealed statistically significant differences between the study groups. Group 1 showed autonomic dysfunction, which altered gastric myoelectrical activity. Bradygastria was 46.5%, but in 5 patients came to 65–70%. Another sign of altered myoelectrical activity in this group was a significantly higher amplitude of ECA waves.

In group 2, elevated ICP was manifested by headache, vomiting, bradycardia 40–60 bpm, somnolence, bradypsychia and motor sluggishness, and in four patients by papilloedema. The GCS score in these patients was 9–15.

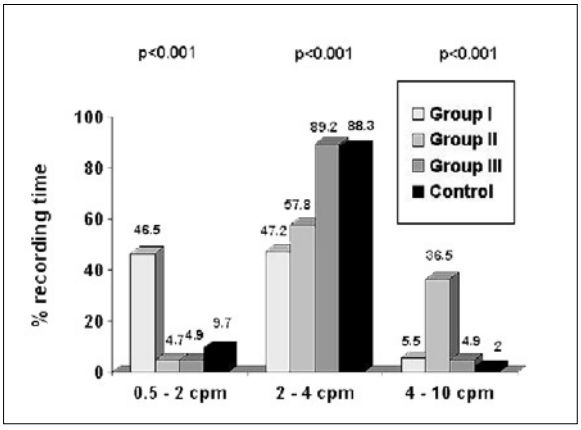

Figure 4. Gastric myoelectrical activity in the study groups.

Table 9. GCS score in group 2, depending on the presence or absence of papilloedema.

Papilloedema	N	GCS		P
		x±SD	min–max	
Absent	12	13±1	12–14	NS
Present	4	13±1.5	10–15	

In 12 patients in this group, tachygastria up to 9 cycles/minute was observed. The percentage of tachygastria was 36.5. In the 4 patients with papilloedema and imminent subtentorial invagination, 56% bradygastria was observed.

In group 3, with a slightly shifted cerebral midline on brain CT (4–8 mm), dysrhythmia and tachygastria below 15% were observed.

Discussion

ICP pressure over 20 cm H_2O due to TBI [1–4] and neoplastic growth within the central nervous system leads to autonomic dysfunction [5], which in turn alters internal organ function, also affecting the gastrointestinal system. Ott et al. studied 12 patients after severe and moderate TBI (GCS 4–10) and observed delayed gastric emptying [6]. These patients also displayed an abnormal biphasic response: gastric emptying faster than normal during the early stage, but prolonged later. In all patients with initially delayed emptying, ICP was also elevated.

Brodie [7] and Watanabe [8], using simple techniques in rats, observed in their studies that gastric emptying increased after stress. Koo et al.[9] found out that cold-restraint stress delayed gastric emptying in rats. Garrick et al. [10] demonstrated that elevated ICP decreases the amplitude of gastric and duodenal contractions by over 80% and 60%, respectively. Bethanechol reversed the inhibited gastric and duodenal contractility, but only for 7.3±0.6 min. Normal gastric motility returned after the reduction of ICP. Rimmer [11] found that increased ICP in patients with brain tumors markedly delays gastric emptying. However, the exact mechanism of this relationship is not clear. Wood and Camilleri [12] reported upper gut motility disorders in a patient with a mild tumor of the medulla (astrocytoma grade II), which developed 7 months before neurological symptoms. Haig et al. [13], in a 2.5-year follow-up study of a patient after severe head injury, found gastric motility disorders. Pathomorphological examinations revealed injury to the pons and medulla, and microscopy showed that it was located in the dorsal nucleus and vagal nerve. Thompson et al. [14] demonstrated that cold stress significantly delayed gastric emptying in humans. Van Miert and De La Parra [15] found that endotoxins and lipopolysaccharides are strong inhibitors of gastric emptying in rats.

Table 8. Gastric myoelectrical activity in group 2, depending on the presence or absence of papilloedema.

Frequency of slow waves	Papilloedema	n	% recording time	P
			x±SD min–max	
0.5–2 cpm	Absent	12	4.7±7.1 (0–23.1)	p=0.004
	Present	4	51.1±9.7 (40–63)	
2–4 cpm	Absent	12	57.8±12.1 (33.3–74.1)	NS
	Present	4	42.3±12.6 (30–60)	
4–10 cpm	Absent	12	36.5±112.1 (22.2–66.7)	p=0.004
	Present	4	6.5±4.9 (0–12)	

Endotoxins are known to release interleukin-1 and other cytokines. Nompleggi et al. [16] demonstrated that recombinant interleukin-1, administered intraventricularly, significantly decreases gastric emptying in rats. TBI patients have been found to have increased interleukin-1 levels in the cerebrospinal fluid.

In a study by Chen [17], the percentage of 2–4 cycles/min slow waves was 88.9% in adults, and thus did not differ from our findings in the controls. The almost 20% dysrhythmia in patients over 60 years of age, reported by Lorens [18], is a result of physiological aging of the autonomic system and increased sympathetic activity. Nishimura [19] described tachygastria in 11% of patients over 60 years of age in contrast to an earlier age group, in which tachygastria was less frequent. No significant differences in gastric myoelectrical activity in the interprandial period were seen between men and women in the control group.

In the present study, altered gastric electrical activity was found in group 1, consisting of TBI patients with elevated ICP grade III, detected in brain CT from the presence of a prominent shift of the cerebral midline over 12 mm, invisible subarachnoid cisterns on the cerebral vault, and cisterns at the base. In this group with severe head injury, 22 patients (91.7%) showed bradygastria of 46.5%, which was significantly different from bradygastria in group 2

Table 10. Gastric myoelectrical activity in the study groups with elevated ICP.

Group	n	Predominance of							
		Bradygastria		Normogastria		Tachygastria		Dysrrhythmia	
I	24	22	*(91.7%)*	0	–	2	*(8.3%)*	0	–
II	16	4*	*(25%)*	0	–	12	*(75%)*	0	–
III	10	1	*(10%)*	8	*(80%)*	0	–	1	*(10%)*

* 4 patients with papilloedema

$Chi^2 = 67.121$ $p < 0.001$

(16.3%), group 3 (4.9%) and the controls (9.7%). Only two patients (8.3%) in group 1 with elevated ICP grade II, in whom the midcerebral line shift was 9–10 mm, were found to have tachygastria. In group 2, consisting of patients with neoplastic growth within the central nervous system, tachygastria of 36.5% prevailed in 75% of the patients. In patients with papilloedema and subtentorial invagination, bradygastria of 56.1% was observed.

We may assume that the increasing symptoms of high ICP stimulate gastric myoelectrical activity during the early stage, which is then decreased in the upper gut due to intracranial shifts, manifested by prominent bradygastria and delayed gastric emptying, as observed by Ott et al. [14]. Biphasic emptying (faster during the early stage after injury and prolonged later) may be accounted for by altered gastric myoelectrical activity, complicated by increased ICP.

The dynamically developing symptoms of increased ICP in patients with rapidly expanding intracranial hematomas or brain tumor hemorrhages produce bradygastria of 46%. In patients with the symptoms of elevated ICP, the amplitude of ECA was found to be higher than in healthy controls.

CONCLUSIONS

1. The greatest changes in gastric myoelectrical activity were found in patients with elevated ICP and coma (GCS score 4–7) due to head injury. Bradygastria occurred in 46.5% of the recording.

2. Major electrogastrographic changes were seen in patients with neoplastic growth in the central nervous system: dysrrhythmias, sometimes as high as 36.5%.

3. Slowly developing symptoms of increased ICP during the early stage alter gastric myoelectrical activity, which is manifest by increased frequency of electrical rhythm and signal amplitude.

4. Rapidly developing symptoms of increased ICP in patients with imminent subtentorial invagination decrease the electrical rhythm of the stomach and increase the signal amplitude.

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